

**U.S. Department of Labor**

Office of Administrative Law Judges  
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**Issue Date: 15 November 2005**

CASE NO.: 2004-BLA-6798

In the Matter of:

DORIS J. OSOBA, Widow of  
CHARLES OSOBA  
Claimant

v.

BETHENERGY MINES, INC.  
Employer

and

DIRECTOR, OFFICE OF WORKERS'  
COMPENSATION PROGRAMS  
Party in Interest

**APPEARANCES:**

Debra L. Henry, Esq.  
For the Claimant

John J. Bagnato, Esq.  
For the Employer

Before: DANIEL L. LELAND  
Administrative Law Judge

**DECISION AND ORDER - AWARDING BENEFITS**

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 et seq. (Act). In accordance with the Act and the pertinent regulations, this case was referred to the Office of Administrative Law Judges by the Director, Office of Workers' Compensation Programs for a formal hearing.

Benefits under the Act are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis or to the survivors of persons whose death was caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lungs arising from coal mine employment and is commonly known as black lung.

A formal hearing was held in Pittsburgh, Pennsylvania on June 22, 2005, at which all parties were afforded full opportunity to present evidence and argument, as provided in the Act and the regulations found in Title 20 Code of Federal Regulations. Regulation section numbers mentioned in this Decision and Order refer to sections of that Title. At the hearing, Director's exhibits (DX) 1-36, Claimant's exhibits (CX) 1-9, and Employer's exhibits (EX) 1-5 were admitted into evidence. The June 21, 2005 deposition of Dr. Francis H. Y. Green was submitted by Claimant on July 20, 2005, and has been marked as CX 10. Claimant and Employer submitted closing briefs.

### ISSUES

- I. Existence of pneumoconiosis.
- II. Causal relationship of pneumoconiosis and coal mine employment.
- III. Causation of death.

### FINDINGS OF FACT AND CONCLUSIONS OF LAW <sup>1</sup>

#### Procedural History and Background

Doris J. Osoba (Claimant) is the surviving spouse of Charles Osoba (the miner), who was born on September 19, 1924 and died on October 31, 1998. (DX 1, 11). The miner was engaged in coal mine employment for twenty-nine years and one and one half months, ending in 1986. (DX 5-8, 27). He filed a lifetime claim on January 16, 1986 which was denied by the district director on March 7, 1986 because the miner did not meet any of the conditions of entitlement. (DX 1). The miner submitted a request for modification on September 9, 1986 that was denied by the district director on November 24, 1986. (DX 1). Claimant filed a claim for survivor's benefits on May 30, 2003 which was denied by the district director on June 29, 2004. (DX 3, 27). Following Claimant's request for a hearing, this case was referred to the Office of Administrative Law Judges on September 14, 2004 (DX 33).

#### Medical Evidence

##### Chest x-rays

| <u>Exhibit</u> | <u>Date</u> | <u>Physician</u> | <u>Interpretation</u> |
|----------------|-------------|------------------|-----------------------|
| CX 4           | 10/25/1995  | Cappiello, BCR/B | 2/2, q/t              |
| EX 4           | 05/20/1998  | Fino, B          | 0/0                   |
| EX 4           | 05/23/1998  | Fino, B          | 0/0                   |
| CX 3           | 07/31/1998  | Cappiello, BCR/B | 2/2, q/t              |

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<sup>1</sup> The following abbreviations have been used in this opinion: DX=Director's exhibit, CX=Claimant's exhibit, EX=Employer's exhibit, TR=Transcript of hearing, BCR=Board certified radiologist, B=B-reader.

### Hospital and Treatment Records

The records of Dr. Neil J. Hart, the miner's cardiologist since 1985, and Dr. Jesus S. Evangelista, the miner's treating physician, are at DX 16. The records reflect approximately a forty pack year smoking history, ending in 1980. The records show that the miner had an anteroseptal myocardial infarction in October of 1985. On February 15, 1988, Dr. Hart observed coronary atherosclerotic heart disease. On January 30, 1989, he diagnosed coronary atherosclerotic heart disease with angina. The miner underwent a coronary artery bypass grafting times four in 1989. Dr. Hart continued to see the miner over time and assessed his coronary atherosclerotic heart disease as stable. On April 15, 1998, the miner underwent a cardiac catheterization. He was diagnosed as having severe coronary atherosclerotic heart disease. On April 22, 1998, the miner underwent a coronary angioplasty to open an obstruction in the left anterior descending artery. The miner underwent a subsequent successful rotoblator for the same obstruction on May 1, 1998. On June 8, 1998, Dr. Hart observed that the miner had developed congestive heart failure in May while vacationing in Florida, where he was hospitalized. However, Dr. Hart assessed that the miner's coronary atherosclerotic heart disease was stable.

Upon his return to Pennsylvania, the miner was re-hospitalized at The Washington Hospital for pulmonary edema, shortness of breath and weakness. On June 23, 1998, Dr. Evangelista found that the miner suffered from coronary atherosclerotic heart disease, hypertension, and silicosis. He noted that the miner had a history of congestive heart failure, but that it was not evident on the day of his examination. July 14, 1998, Dr. Evangelista's impressions included dyspnea, coronary atherosclerotic heart disease, and silicosis, and documented the miner's two to three day history of increasing shortness of breath. While the miner was admitted to the hospital for congestive heart failure, Dr. Evangelista did not note congestive heart failure among his impressions. On July 31, 1998, his impressions included pulmonary edema with an unknown etiology, and coronary atherosclerotic heart disease, and congestive heart failure was not noted. On September 29, 1998, the miner's chief complaints were increased shortness of breath and dizziness, night sweats, and cough with dark sputum. The miner was using oxygen at home as needed. Dr. Evangelista observed that the miner's history of chronic obstructive pulmonary disease was followed by Dr. Iracki, the miner's pulmonologist. Dr. Evangelista's impressions included coronary atherosclerotic heart disease, chronic obstructive pulmonary disease/emphysema, and silicosis. On October 12, 1998 the miner complained of increasing shortness of breath and was wearing the oxygen mask continuously at home. Impressions included dyspnea, coronary atherosclerotic heart disease, chronic obstructive pulmonary disease/emphysema, and silicosis.

### Autopsy and Medical Reports

Dr. Evangelista completed the death certificate and listed progressive idiopathic interstitial pulmonary fibrosis as the immediate cause of the miner's death. (DX 11). Other significant conditions contributing to the miner's death were congestive heart failure, coronary atherosclerotic heart disease, chronic obstructive lung disease and coal worker's pneumoconiosis. *Id.*

Dr. Cyril H. Wecht, a board certified anatomic, clinical and forensic pathologist, performed an autopsy on the miner on November 3, 1998. (DX 13). Upon gross examination, Dr. Wecht noted that the external surfaces of the lungs showed black anthracotic streakings to a marked degree and uniform patterns of firm, elevated gray-white nodules, ranging from 2 to 4 mm in diameter. Emphysematous blebs were present at the peripheral margins of the upper lobes and right middle lobe, ranging from approximately 2.5 to 4 mm in diameter. The color of the lungs were a light pinkish-tan and a darker reddish-brown to reddish-purple. Sectioning the lungs revealed black anthracotic streakings and a fine, uniform nodular pattern similar to that shown externally, though to a lesser degree. The pulmonary arterial walls appeared to be slightly thickened. The miner's lymph nodes were enlarged and had a black anthracotic appearance on cut sections. The miner's heart showed a few small, patchy areas of gray-white fibrosis in the posterior wall of the left ventricle and interventricular septum in the midportion of the heart. The coronary arteries showed moderate to severe atherosclerosis with calcific debris. The microscopic examination was consistent with the gross examination.

Under the heading of hypertensive and atherosclerotic cardiovascular disease, Dr. Wecht diagnosed the following: (1) status, post-coronary bypass graft surgery (triple vessels); (2) fibrous pericarditis, extensive, secondary to operative procedure; (3) myocardial hypertrophy; (4) atherosclerosis of coronary arteries, moderate to severe; (5) atherosclerosis of aorta, severe, with aneurysms of distal abdominal portion; and (6) myocardial fibrosis, focal, left ventricle. Under the heading of chronic obstructive pulmonary disease, Dr. Wecht diagnosed the following: (1) anthracosilicosis; (coal worker's pneumoconiosis); (2) pulmonary emphysema, bilateral; (3) pulmonary fibrosis, bilateral; (4) fibrohyaline and fibroanthracotic macules and micronodules; (5) fibroanthracosis and fibrohyalinization of mediastinal and peribronchial lymph nodes; (6) fibrous pleuritis, bilateral, focal; (7) cor pulmonale; (8) increased anteroposterior diameter of chest; (9) pulmonary osteoarthropathy; (10) chronic passive congestion of the lungs; and (11) bronchopneumonia.

On December 11, 1998, Dr. Wecht submitted a supplemental report in which he reiterated the relevant autopsy findings. (DX 14). Examination of the lung tissue under polarized light revealed scattered birefringent crystals, consistent with silica. Dr. Wecht opined that the gross and microscopic findings were consistent with a clinical diagnosis of pneumoconiosis. He determined that the miner died as a result of hypertensive and atherosclerotic cardiovascular disease. The coal worker's pneumoconiosis, the basis for the miner's chronic obstructive pulmonary disease, was a substantially contributing factor in the miner's death, Dr. Wecht declared.

On September 18, 1999, the miner's medical records were reviewed by Dr. Gregory J. Fino, a board certified pulmonologist. (DX 17). He stated that in previous examinations of the miner, he diagnosed simple coal worker's pneumoconiosis but no respiratory impairment or pulmonary disability was present. Dr. Fino observed that there was no progression of any occupational disease, but that the miner's cardiac condition had deteriorated. Dr. Fino determined that coal mine dust inhalation did not cause or contribute to the miner's death.

In a report dated November 12, 2003, Dr. Stephen T. Bush, a board certified anatomic and clinical pathologist, reviewed the miner's medical records and autopsy slides. (DX 17). After

reviewing the histologic slides, Dr. Bush concluded that the miner did not have occupationally acquired pulmonary disease. He noted a few tiny sclerotic collagenous pleural nodules measure up to .3 cm, devoid of pigment or birefringent mineral, but he stated that the slides showed no evidence of coal worker's macules, micronodules or macronodules. Dr. Bush stated that significant dust pigment would have discolored the lungs black over substantial areas, as opposed to the pink and red color of the lungs Dr. Wecht observed at autopsy. Polarized light examination revealed rare birefringent silica. Dr. Bush found that no fibrous reaction was associated with the deposition of pigment, also found in the lymph node in extremely small quantity. He stated that the fibrosis in the lung consisted of small, patchy scars. The lungs showed a moderate degree of centrilobular emphysema in all areas. Dr. Bush stated that the emphysema was unrelated to the dust pigment or birefringent mineral deposits and was typical of emphysema from smoking. The lungs also showed acute bronchopneumonia.

Dr. Bush concluded that the cause of death was severe cardiac disease resulting from severe atherosclerotic coronary artery disease, previous myocardial infarction and hypertensive myocardial disease, resulting in significant myocardial hypertrophy. Bronchopneumonia was a probable contributor to death. Dr. Bush found no causal relationship between occupational dust exposure and death, stating that the dust pigment present in the miner's lungs produced no impairment of lung function. He further found that there was no evidence to support a diagnosis of cor pulmonale, and that the changes in the miner's heart size were a result of hypertension. He commented on the miner's September 26, 1998 hospitalization, wherein the miner's prescription of amiodarone, used to treat cardiac rhythm disturbance, was reduced, as it was thought to be contributing to the miner's pulmonary symptoms.

In a report dated March 31, 2005, Dr. Francis H. Y. Green, a board certified anatomic pathologist, reviewed the miner's medical records and autopsy slides. (CX 7). Dr. Green's examination of the autopsy slides revealed the presence of moderately severe pneumoconiosis characterized by the presence of silicotic nodules, mostly subpleural in location, micronodules scattered throughout pulmonary parenchyma, and irregular interstitial fibrosis in which dust particles were present. Silicotic nodules were also seen in the lymph nodes. Dust within these lesions revealed particles consistent with coal dust, together with birefringent particles, consistent with silicates and silica morphology. Dr. Green attached microphotography to illustrate lesions in the miner representative of pneumoconiosis. He also observed scar emphysema. Dr. Green noted evidence of necrotizing bronchopneumonia and chronic congestive heart failure. He found no evidence of amiodarone toxicity, indicating that the medication did not contribute to the miner's pulmonary condition. Section of the miner's left ventricle showed patchy areas of fibrosis consistent with the history of old myocardial infarction. There was evidence of fiber hypertrophy but no evidence of acute myocardial infarction. Coronary arteries showed moderately severe coronary artery atherosclerosis but without evidence of thrombosis. Dr. Green diagnosed the following: (1) moderately severe to severe simple coal worker's pneumoconiosis comprising silicotic nodules, coal dust micronodules, and irregular interstitial fibrosis with dust; (2) silicotic nodules within the lymph nodes; (3) focal, scar and centriacinar emphysema; (4) necrotizing bronchopneumonia associated with vascular thromboses; (5) chronic congestive heart failure; (6) cardiac hypertrophy; (7) myocardial infarction; and (8) coronary artery atherosclerosis.

Dr. Green concurred with Dr. Wecht's microscopic diagnoses. Dr. Green concluded that the immediate cause of the miner's death was bronchopneumonia with the underlying cause of death being pneumoconiosis. Severe hypertensive/ischemic cardiovascular disease was a significant contributing factor to death. In making this determination, Dr. Green relied on the stability of the miner's cardiac condition before death, citing no evidence of an acute cardiac event such as myocardial infarction or coronary artery thrombosis at autopsy, and noting, by contrast, a clinical progression of his lung disease shortly before his death. Dr. Green determined that coal dust as well as cigarette smoking would account to some extent for the miner's emphysema. However, cigarette smoking could not account for the miner's restrictive lung disease and the irregular opacities seen on the x-rays. The basis for the irregular opacities was apparent at autopsy which showed irregular bands of interstitial fibrosis.

Dr. Green stated that silicas, as well as the corticosteroids prescribed to the miner to treat the idiopathic pulmonary fibrosis, are known to suppress the immune system, and could have made the miner more vulnerable to pneumonia. Thus, he concluded that respiratory failure due to bronchopneumonia would be causally related to treatment for the underlying pneumoconiosis. Further, the miner's acute lung disease (bronchopneumonia), together with his chronic lung disease (pneumoconiosis and emphysema), increased the work of the heart leading to cardiac failure, the cause of death. Dr. Green concurred with Dr. Wecht's diagnosis of cor pulmonale, and disagreed with Drs. Bush and Hurwitz that right heart hypertrophy was due exclusively to heart disease. Dr. Green opined that the most reasonable assessment of the miner's right heart hypertrophy was that the condition was attributable to his hypertensive and ischemic heart disease, as well as the underlying lung disease.

The miner's medical records were also reviewed by Dr. Larry E. Hurwitz, a board certified cardiologist, who prepared a report on January 26, 2004. (EX 1). Dr. Hurwitz noted that at autopsy, the heart weighed 675 grams, whereas 350 grams is normal. Significant fibrous adhesions surrounded the myocardium. The coronary arteries demonstrated moderate to severe occlusive disease. The aorta demonstrated severe atherosclerotic disease. There was also evidence of myocardial fibrosis indicative of previous myocardial infarction. Dr. Hurwitz disagreed with Dr. Wecht's diagnosis of cor pulmonale. The miner had evidence of significant hypertensive cardiovascular disease with mild pulmonary hypertension. The physical examinations over his lifetime did not demonstrate clinical findings of right ventricular dysfunction. Rather, the miner's clinical course was characterized by left ventricular dysfunction with recurrent pulmonary edema. Further, he stated that in a setting of significant hypertensive heart disease and recurrent left ventricular failure, such as this one, it is impossible to diagnose cor pulmonale. Dr. Hurwitz concluded that the miner's death occurred as a result of developing bronchopneumonia, in the setting of progressive heart failure. Exposure to coal dust played no role in the miner's death.

Dr. Daniel A. Iracki, the miner's pulmonologist since 1987, prepared a report on February 27, 2004. (CX 1). Dr. Iracki stated that when he first examined the miner in July of 1987, he concluded that the miner had coal worker's pneumoconiosis based on the miner's history, symptoms, and chest x-rays. No specific treatment was prescribed for the disease. He stated that he saw the miner again in May of 1988 for recurrent hemoptysis, and did not see the miner again until 1997. Dr. Iracki saw the miner almost daily in October, 1998. He found that the

miner died from cardiovascular failure. He opined that pneumoconiosis was a substantial contributing cause of the miner's death, in that pneumoconiosis caused the miner's pulmonary fibrosis, which worsened the miner's overall cardiac function. He noted that there was no evidence at autopsy of acute myocardial infarction, pulmonary embolism, cerebrovascular infarction or aneurysmal rupture. The pulmonary fibrosis diminished the miner's capacity to recover from the cardiac and pulmonary distress that led to his death in 1998.

Dr. Fino submitted a supplemental report dated November 21, 2004 (EX 2). Dr. Fino stated that his current and previous readings of the x-rays were negative for pneumoconiosis. In response to a number of positive x-ray readings, as well as a number of diagnoses of pneumoconiosis, he asserted that any radiographic progression was due to the miner's heart condition, or reflective of old granulomatous disease. Dr. Fino stated that the miner's normal blood gas studies and spirometries demonstrated that there was no evidence of ventilatory impairment and that the miner had no functional lung disease prior to his death. Therefore, lung disease could not have contributed to his death. Even if he assumed pneumoconiosis was present, it neither caused nor contributed to any respiratory impairment or pulmonary disability, nor did it cause or hasten the miner's death. Dr. Fino stated cause of death was significant coronary artery disease, unrelated to the inhalation of coal dust. Dr. Fino also referred to medical literature to support the proposition that pneumoconiosis does not increase the incidence of coronary artery disease, sudden death due to heart disease, or sudden cardiac arrest.

In Dr. Bush's December 2, 2004 deposition, he reaffirmed his position that coal worker's pneumoconiosis was not present. (EX 3 at 15, 17). He stated that the miner's shortness of breath was related to congestive heart failure, superimposed on lungs affected by cigarette smoking. (*Id.* at 18). Dr. Bush noted that the miner had emphysema from smoking and was not occupationally related. (*Id.* at 18, 34-35). Dr. Bush stated that because of the limited extent of pulmonary disease in the lungs, there was no effect on the heart. (*Id.* at 34). He further stated that less than fifty percent damage to lung tissue would have no adverse effect on the heart pumping blood. (*Id.* at 41). He explained that any right ventricular thickening in the miner's heart was secondary to left-sided thickening. (*Id.* at 20-21). He testified that any radiological changes suggestive of pulmonary fibrosis were in fact due to pneumonia and blood clots. (*Id.* at 23).

In Dr. Fino's January 3, 2005 deposition, he testified that despite radiographical and pathological assessments of pneumoconiosis, his negative finding for pneumoconiosis would not change, as pulmonary impairment is assessed by valid lung function studies, not specific diagnoses. (EX 4 at 22). Assuming the presence of pneumoconiosis, Dr. Fino maintained that the miner's lung function was not impaired. He testified that lung disease could only cause or contribute to the miner's death if it was causing impairment prior to death, which in this miner's case, it was not. (*Id.* 31-32). Dr. Fino based this conclusion on the miner's normal pulmonary function studies, his on-going treatment for heart disease, and an absence of treatment for primary lung disease during his life. (*Id.* at 43). Dr. Fino testified that an impairment must be evidenced by pulmonary function studies in order for a pulmonary condition to be related to death. (*Id.* at 45). Dr. Fino agreed with Dr. Hurwitz's assessment that cause of death was bronchopneumonia; and no chronic lung condition contributed to his death. (*Id.* at 53).

Dr. Hurwitz was deposed on April 14, 2005. (EX 5). Dr. Hurwitz observed that pulmonary disease did not play a role in the care or treatment of the miner's heart condition over the miner's lifetime. (*Id.* at 15). He opined that none of the clinical signs of cor pulmonale were present. (*Id.* at 17-18). He stated that he would accept a pathologist's interpretation of the miner's lung condition, but that any existing lung condition had no effect on the miner's treatment, care or his history of heart disease. (*Id.* at 22-23, 34-35). Dr. Hurwitz testified that the presence of opacities or fibrosis would affect the transfer or process of oxygen only if it was shown to be functionally significant, which it was not in this case. (*Id.* 25-26). In fact, Dr. Hurwitz opined that some fibrotic changes could be attributed to those times that the miner was in congestive heart failure. (*Id.* at 26-27). Dr. Hurwitz testified that the miner's heart muscle was not getting appropriate oxygenation, due, in part, to his arteries, but primarily due to his heart attack in 1985. (*Id.* 29-32). Despite improving cardiac condition as to specific surgeries, the cardiac history of the miner overall was one of a severely diseased heart. (*Id.* at 28).

In a supplemental report dated April 20, 2005, Dr. Iracki agreed with Dr. Fino that the miner's pneumoconiosis did not cause his arteriosclerotic heart disease, but disagreed with Dr. Fino's conclusion that the miner did not have pneumoconiosis or that it was not a substantial contributing cause of his death. (CX 9). Dr. Iracki concluded, on the basis of the miner's work history, x-rays, CT scans, and the autopsy findings, that pneumoconiosis was present. Dr. Iracki stated that the only evidence against a finding of pneumoconiosis were the miner's pulmonary function studies. However, based on the aggressive treatment for heart failure, the miner's history of intermittent hemoptysis, and in light of the autopsy findings, the pulmonary fibrosis and emphysema caused by pneumoconiosis were a substantial contributing cause of death.

Dr. Green was deposed on June 21, 2005 (CX 10). He stated that idiopathic fibrosis was not found at autopsy; however, the autopsy revealed that the fibrosis was due to coal mine dust. (*Id.* at 21). Dr. Green reiterated that the silicotic nodules, coal dust micronodules, and interstitial fibrosis associated with dust fulfill the criteria for pneumoconiosis. (*Id.* at 8). The miner's fibrosis was attributable to employment because silicotic nodules are due to the presence of silica in coal mine dust. (*Id.* at 10). The fibrosis increased the work load on the heart to oxygenate the blood, raising pressure on the right ventricle giving rise to pulmonary hypertension/cor pulmonale. (*Id.* at 17) Dr. Green agreed with Dr. Fino that lifetime pulmonary function testing was relatively normal/clinically insignificant. Dr. Green testified that in while there was no evidence of impairment from the pulmonary function studies, there was evidence of restrictive and obstructive deficits that operated to cancel each other out in the objective testing. (*Id.* at 19, 56). Dr. Green concluded that the miner's smoking and coal mine employment histories were each equally responsible for his emphysema. (*Id.* at 22-23). He further testified that smoking does not cause significant lung fibrosis. (*Id.*) Dr. Green added that pneumoconiosis hindered the miner from recovering from pneumonia because it can suppress the immune system and the ability to fight off infection. (*Id.* at 27-28).

### Conclusions of Law

As a preliminary matter, I will address a pending issue regarding the admissibility of the parties' autopsy evidence. § 725.414 sets forth limitations on documentary medical evidence. It states, in part, that each party may submit one autopsy report as part of its affirmative case, one

autopsy report in rebuttal of the opponent's case, and when a report is the subject of rebuttal, the party submitting the original report may submit an additional statement from the authoring physician. § 725.414(a)(2)-(3).

In *Kalist v. Buckeye Coal Co.*, BRB No. 03-0743 BLA (July 23, 2004) (unpub.), the Benefits Review Board (Board) held that a report by a physician conducting an examination of a body post-mortem constitutes the autopsy report of record. *Kalist* at 3. The Board noted that the report of the physician in *Kalist* who "only reviewed medical records and autopsy slides" would be considered the report of a reviewing physician. *Id.*

Applying *Kalist* to the case at bar, Dr. Wecht, the autopsy prosector, performed the only examination of the miner's body post-mortem. As such, Dr. Wecht's report is the autopsy report of record, and was admitted as part of Claimant's affirmative case. Employer did not have a physician present at, or participate in, the autopsy conducted by Dr. Wecht, thus, Employer does not have an autopsy report as part of its affirmative case. *Kalist* at 4. Therefore, as I previously ruled, the report of Dr. Bush was properly admitted as Employer's rebuttal to the autopsy report. The report of Dr. Naeye was properly excluded, as it would exceed the quotient so provided by § 725.414, either as a second rebuttal to the autopsy report, or as a third medical report. The report of Dr. Green was properly admitted as one of Claimant's two medical reports.

#### Existence of Pneumoconiosis

Benefits are provided to eligible survivors of a miner whose death is due to pneumoconiosis. § 718.205(a).<sup>2</sup> The Board has held that, in a survivor's claim, the administrative law judge must make a threshold determination as to the existence of pneumoconiosis under § 718.202(a) prior to considering whether the miner's death is due to the disease. *Trumbo v. Reading Anthracite Co.*, 17 BLR 1-85 (1993).

The record includes four interpretations of four x-rays; two are positive for pneumoconiosis and two are negative for pneumoconiosis. The chest x-ray interpretations of physicians who are both board certified radiologists and B readers are entitled to the greatest weight. *Cranor v. Peabody Coal Co.*, 22 BLR 1-1 (1999) (en banc on recon.). Dr. Cappiello is a dually qualified physician. (CX 5). Dr. Fino is a B-reader. (EX 2). The Board has held that it is proper to credit the interpretation of a dually qualified physician over the interpretation a B-reader. *Cranor*, *supra*. Dr. Fino reviewed two May, 1998 x-rays taken within three days of each other as negative for pneumoconiosis. Dr. Cappiello re-read the October 25, 1995 x-ray and the July 31, 1998 x-ray, interpreting each x-ray as positive for pneumoconiosis. Because Dr. Cappiello is a dually qualified physician, I accord the x-ray interpretations of Dr. Cappiello greater weight. Therefore, a preponderance of the chest x-ray evidence establishes clinical pneumoconiosis.

Clinical pneumoconiosis encapsulates only a small number of compensable afflictions under the Act. *Barber v. Director, OWCP*, 43 F.3d 899, 901 (4th Cir. 1995). An administrative

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<sup>2</sup> Pneumoconiosis is defined as a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment, and it includes both medical, or clinical, pneumoconiosis and statutory, or legal pneumoconiosis.

law judge should review the evidence in light of the broader, regulatory definition of that which may comprise the disease.<sup>3</sup>

Initially, I find that the opinions of the three pathologists in this case, Dr. Bush, Dr. Green and Dr. Wecht, are entitled to more weight than the opinions of the pulmonologists, Dr. Fino and Dr. Iracki, and the cardiologist, Dr. Hurwitz. Pathologists are trained to make clinical judgments regarding the degree of pulmonary impairment and disability.<sup>4</sup> Dr. Green diagnosed moderately severe to severe simple coal worker's pneumoconiosis, irregular interstitial fibrosis with dust, silicotic nodules within the lymph nodes and emphysema. Dr. Wecht diagnosed coal worker's pneumoconiosis, pulmonary emphysema and pulmonary fibrosis. He found gross and microscopic evidence of pneumoconiosis, and identified nodules measuring 2-4 mm in diameter. I find that the diagnoses of Drs. Green and Wecht are well-documented and well-reasoned. Therefore, I find that the opinions of Drs. Green and Wecht are entitled to great weight.

Dr. Bush found no evidence of coal worker's macules, micronodules or macronodules. He provided reasons for why the radiological evidence gave the appearance of pneumoconiosis if in fact, the disease was not present. However, he offered no explanation for the nature of the tiny sclerotic collagenous pleural nodules he observed, nodules that Dr. Green testified were classic pneumoconiotic nodules. Nor did Dr. Bush offer an explanation for what Drs. Wecht and Green observed as pneumoconiotic lesions at autopsy. Also, while Dr. Bush testified that the miner did not have idiopathic fibrosis, he did not offer an opinion as to the etiology of the fibrosis that he noted in his report. Further, Dr. Bush admitted on cross examination that his findings in reference to the presence of an occupationally acquired pulmonary disease pertained only to the medical, and not legal definition of pneumoconiosis. (EX 3 at 28-30). Legal pneumoconiosis includes emphysema and chronic obstructive pulmonary disease, if related to coal mine employment. § 718.201(a)(2) and (b); *Hughes v. Clinchfield Coal Co.*, 21 BLR 1-134, 1-139 (1999). Dr. Bush diagnosed emphysema and attributed the disease to smoking. Dr. Bush did not adequately justify why the miner's employment history could be excluded as a contributing factor to the disease, stating only that coal dust did not cause the type of emphysema he observed in the miner's lungs. In light of the foregoing, Dr. Bush's opinion is not well-reasoned and I accord it little weight. After weighing all of the autopsy evidence, I find that Claimant has established the existence of clinical, as well as legal, pneumoconiosis.

The record contains the opinions of three physicians. Dr. Hurwitz, a cardiologist, deferred to the pulmonologists for diagnosis of the miner's pulmonary condition. Thus, Dr. Hurwitz's opinion is not probative on this issue. In his 1999 report, Dr. Fino diagnosed that the miner had simple coal worker's pneumoconiosis. However, in his 2004 report, Dr. Fino found no evidence of pneumoconiosis. He based this opinion on the radiological evidence and a lack of treatment for lung disease during the miner's lifetime. Dr. Fino's contradictory diagnoses were not resolved at deposition or by subsequent report. Additionally, Dr. Fino also relied to some extent

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<sup>3</sup> Legal pneumoconiosis "includes any chronic lung disease or impairment... arising out of coal mine employment. This definition includes, but is not limited to, any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment," which includes "any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment." § 718.201.

<sup>4</sup> Autopsy evidence is the most reliable evidence of the existence of pneumoconiosis. *Terlip v. Director, OWCP*, 8 BLR 1-363 (1985).

on evidence not of record, including evidence from the lifetime claim.<sup>5</sup> Evidence of the miner's pulmonary condition over his lifetime is hardly conclusive of the miner's pulmonary condition at the time of his death. Therefore, I accord Dr. Fino's opinion little weight. Dr. Iracki diagnosed coal worker's pneumoconiosis, but only generally referred to the medical evidence that he reviewed in conjunction with his diagnosis. However, Dr. Iracki was the miner's treating physician, and his treatment history and relationship with the miner is entitled to some deference under the Act.<sup>6</sup> Furthermore, his diagnosis is supported by the weight of the evidence. Therefore, I accord the opinion of Dr. Iracki greater weight than that of Dr. Fino. After weighing all of the physician opinion evidence, I find that Claimant has established the existence of pneumoconiosis.

Therefore, after weighing all of the evidence together, I find that Claimant has established the existence of clinical, as well as legal pneumoconiosis.

### Causal Relationship

Claimant is entitled to the presumption in § 718.203(b) that the miner's pneumoconiosis arose out of coal mine employment because of his twenty nine years and one and one half months of coal mine employment. This presumption has not been rebutted.

### Death Due to Pneumoconiosis

In claims filed on or after January 1, 1982, death will be considered due to pneumoconiosis: (1) where competent medical evidence establishes that the miner's death was due to pneumoconiosis; or (2) where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by complications of pneumoconiosis; or (3) where the presumption in § 718.304 is applicable. § 718.205(c). Pneumoconiosis is a substantially contributing cause of death if it hastened the miner's death. § 718.205(c)(5); *See also Lukosevich v. Director, OWCP*, 888 F.2d 1001 (3d Cir.1989).<sup>7</sup>

An unsupported medical conclusion is not a reasoned diagnosis. *Fuller v. Gibraltar Corp.*, 6 BLR 1-1292 (1984). In his report, Dr. Wecht offered no more than a conclusory

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<sup>5</sup> In *Church v. Kentland-Elkhorn Coal Corp.*, BRB Nos. 04-0617 BLA and 04-0617 BLA-A (Apr. 8, 2005) (unpub.), the Benefits Review Board held that medical evidence submitted in a living miner's claim is not automatically admissible in a survivor's claim filed after January 19, 2001. Evidence in the living miner's claim cannot be considered in the survivor's claim unless it meets the limitations at 20 C.F.R. § 725.414, and the survivor's claim should consider only evidence properly admitted thereunder.

<sup>6</sup> § 718.104(d).

<sup>7</sup> Employer urges that this court apply the standard adopted by the Court of Appeals for the Sixth Circuit in *Eastover Mining Co. v. Williams*, 338 F.3d 501, 517-18 (6th Cir. 2003), wherein the court held, in part, that "legal pneumoconiosis only 'hastens' a death if it does so through a specifically defined process that reduces the miner's life by as estimable time." The Court of Appeals for the Third Circuit recently confronted this issue in *Inland Steel Co. v. Director, OWCP*, 2005 WL 2713691, at \*2 (3d Cir. Oct. 24, 2005). The court found that the *Eastover Mining* standard was inconsistent with its precedents, stating that such an approach would "unnecessarily and improperly raise the burden on a claimant to produce evidence to a medical degree of certainty not previously demanded in this Circuit." *Inland Steel*, 2005 WL 2713691, at \*3. Therefore, Employer's argument is rejected as it directly contravenes the law of the Circuit applicable to this case.

statement that the miner's pneumoconiosis, the basis for his chronic obstructive pulmonary disease, was a substantial contributing factor in his death. Therefore, I find that Dr. Wecht's opinion is not reasoned, and I accord it little weight. Dr. Iracki stated that the miner's "underlying pulmonary fibrosis substantially diminished his capacity to recover from the cardiac and pulmonary insult that led to his death." However, Dr. Iracki offers no explanation as to how the pneumoconiosis specifically impacted the miner's cardiac and pulmonary conditions. Therefore, I find that Dr. Iracki's opinion is not reasoned, and I accord it little weight.

The Court of Appeals for the Fourth Circuit has held that absent "specific and persuasive" reasons, an ALJ may not credit a medical opinion stating that a claimant did not suffer from pneumoconiosis causing respiratory disability after the ALJ accepted the presence of pneumoconiosis. *Scott v. Mason Coal Co.*, 289 F.3d 263, 269 (4th Cir. 2002). In *Soubik v. Director, OWCP*, 366 F.3d 226, 234 (3d Cir. 2004), the Court of Appeals for the Third Circuit confronted a similar set of facts. The physician in *Soubik* concluded that there was no evidence of pneumoconiosis, but advanced an opinion as to cause of death under the assumption that pneumoconiosis *was* present. The Circuit court held that use of such a "superficial hypothetical" did not reconcile the physician's opinion with the ALJ's determination that pneumoconiosis was present.

As previously stated, in direction contradiction to his own findings in 1999, Dr. Fino found no evidence of pneumoconiosis in 2004. Dr. Fino proceeded, however, by assuming that the miner had legal pneumoconiosis for the purposes of determining the cause of death. Dr. Fino's assumption that the miner suffered from legal pneumoconiosis indicates Dr. Fino did not, in fact, diagnose that condition in the miner. Thus, Dr. Fino's assumption constitutes just the sort of superficial hypothetical with which the Third Circuit took issue. Dr. Fino's opinion cannot be reconciled with my determination that the miner had pneumoconiosis. Therefore, I accord the opinion of Dr. Fino no weight. Even if Dr. Fino's contradictory diagnoses did not wholly discredit his testimony, his opinion would be accorded only minimal weight. Dr. Fino testified that it is necessary to have impairment evidenced by pulmonary function studies in order for a pulmonary condition to be related to death. (EX 4 at 45). This is incorrect. Rather, lifetime disability or impairment is not an element of proof in "hastening," and an ALJ may reasonably discount the evidence of those experts who relied heavily on lifetime studies. *Consolidation Coal Co. v. Kramer*, 305 F.3d 203, 210 (3d Cir. 2002).

Dr. Hurwitz declined to assess the miner's pulmonary condition, instead crediting the pulmonologists with ascertaining the presence or absence of pneumoconiosis. Dr. Hurwitz acknowledged that the miner had an underlying pulmonary disease, but he went on to state that any existing lung disease did not affect the miner's heart condition, and it did not contribute to the miner's death. As Dr. Hurwitz neither diagnosed, nor concluded that the miner had pneumoconiosis, it simply does not follow that Dr. Hurwitz could reasonably discount the effect of pulmonary disease on death with such certainty. Thus, Dr. Hurwitz's opinion is accorded no weight.

Dr. Bush evaluated the miner's records only for medical pneumoconiosis.<sup>8</sup> Dr. Bush stated that the miner's fibrosis was not idiopathic, but he did not address the etiology of the fibrosis. Dr. Green pointed out that indeed, the autopsy demonstrated that the miner's fibrosis was not idiopathic; rather, that any fibrosis present was associated with dust deposition together with silicates and silica. Dr. Bush found no causal relationship between occupational dust exposure and death, stating that the dust pigment present in the miner's lungs produced no impairment of lung function. Dr. Bush testified that over fifty percent of the vessels in the lung need to be damaged before lung disease could contribute to the work of the heart. (EX 3 at 40-41). This conclusion is unsupported by any citation to the medical literature. Moreover, Dr. Bush stated that the miner's emphysema was due to smoking, but did not explain why the miner's coal mine employment did not contribute to the disease. Therefore, I find that the opinion of Dr. Bush is not well-reasoned and I accord it little weight.

Dr. Green's opinion and testimony is the most thorough and comprehensive evaluation of the significant findings and non-findings related to the miner's condition and death. He is also the most qualified physician of record. Dr. Green's accomplishments include co-authoring and editing *The Pathology of Occupational Lung Disease*, one of the foremost textbooks on occupational diseases, as well as an eight year tenure as the Chief of the Pathology Section of the National Institute for Occupational Safety and Health (NIOSH). Dr. Green provided two alternative bases to illustrate how pneumoconiosis was a substantial contributing factor in the miner's death. First, the miner's pulmonary fibrosis increased the work of the heart to oxygenate the blood. Dr. Hurwitz testified that the condition of the miner's arteries contributed to the miner's heart muscle not getting appropriate oxygenation. Dr. Green identified that the miner's arteries were patent two weeks before his death, and were thus not a contributing cause of the reduction in oxygenation. Further, the miner's heart condition was well maintained prior to his death. Second, the miner's pulmonary condition weakened his immune system. Dr. Green stated that the corticosteroids prescribed to the miner to treat pulmonary fibrosis are known to suppress the immune system, and could have made the miner more vulnerable to pneumonia. Additionally, the presence of silica can suppress the immune system and the ability to fight off infection. Dr. Fino referred to medical literature in an effort to contradict this conclusion, but his opinion has been discredited by this court. Therefore, I credit the opinion of Dr. Green, as it is better reasoned and better supported than the other physicians of record. After weighing all of the evidence, I find that coal worker's pneumoconiosis was a substantially contributing cause of the miner's death.

The evidence proves that the miner died due to pneumoconiosis. Benefits will be awarded as of October 1, 1998, the first day of the month in which the miner died. § 725.503(c). Claimant's counsel has thirty days to file a fully supported fee application and her attention is directed to §§ 725.365 and 725.366. Employer's counsel has twenty days to respond with objections.

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<sup>8</sup> It is important to note that the court in *Scott* found that a doctor's opinion that a claimant did not have coal worker's pneumoconiosis did not necessarily contradict the ALJ's finding that the claimant had legal pneumoconiosis, and the ALJ could properly rely the opinion of that doctor on causation. *Soubik*, 366 F.3d at 269.

ORDER

IT IS ORDERED THAT Bethenergy Mines, Inc. pay Claimant all the benefits to which she is entitled beginning as of October 1, 1998.

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DANIEL L. LELAND  
Administrative Law Judge

**NOTICE OF APPEAL RIGHTS:** If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Donald S. Shire, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).